January 2, 1953

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Dear Lu:

I just had a letter from Benzer about a "UV conference" at Purdue January 25-26. I want to ask whether you are planning to go (and if so, who with you). The reason is that we will be driving en route to Chamblee, Ga. (via New Orleans, perhaps) and had planned to stop over at Urbana on the 25th or 26th. Spicer will be accompanying Esther and me. There are a few new developments on lysogenization/transduction I wanted to discuss with you and Joe.

We did an X-ray experiment, but 200,000 r (in broth) gave only one decade of attenuation of plaque-formation, and less than that of FA, so this does not seem very promising. You mentioned something about imitating the indirect effects with peroxide. Also, I know there must be somewhere a more detailed account of phage-inactivation with mustard, but I cannot find these offhand. With UV, lysogenization can be completely separated from transduction, and I have so far no evidence of having had any access at all to the included genetic material FA. In view of the assumed locus of indirect X-ray effects, it is rather unlikely that we will get very far with these either, but even the negative information might be useful. [Do you have the supply of reprints of Watson II?]

Some of the mysteries of our lysogenization-transduction correlation may be clearing up. We think that within the progeny of single infected cells transduction occurs in the same clones as are lysogenized. The non-transinduced, phage-sensitive survivors in these experiments are the other progeny of the infected cells. A rather useful tool has come up, a lytic mutant of PLT22, 22V which has the property of being interfered with by 22 itself. Thus, at low multiplicities one can recover as many bacteria resistant to a challenge of 22V ten minutes later as one has added particles of 22. It appears as if all of the transductions are protected against 22V, although only enough PLT22 is added to protect 10% of the total bacteria. This I think may lead to fairly rigorous proof that all transductions involve phage-infected bacteria, which is significant when low multiplicities of infection are involved. As with lambda, infected Salmonella cells give rise to mixed, contaminated colonies. With higher multiplicities of infection, the colonies may be nearly pure lysogenic, but even here there is a suspicion of a residuum of sensitive cells in the "lysogenic" colonies. Under my conditions, I have been getting negigible killing with any multiplicity, though I am fairly confident of Boyd's result in other, more casual expts.

Sincerely,

Joshua Lederberg